[0232] These experiments suggest that copper chelation effectively reduces neointima formation in vivo, and that copper chelation corresponds with a prominent antiproliferative and anti-inflammatory effect. The data disclosed herein also suggest that copper chelation can be a useful tool in the therapeutic management of vascular restenosis.

[0233] The disclosures of each and every patent, patent application, and publication cited herein are hereby incorporated herein by reference in their entirety.

[0234] While this invention has been disclosed with reference to specific embodiments, it is apparent that other embodiments and variations of this invention may be devised by others skilled in the art without departing from the true spirit and scope of the invention. The appended claims are intended to be construed to include all such embodiments and equivalent variations.

1. A method of inhibiting interleukin-1 alpha (IL-lα) release from a cell, said method comprising administering an effective amount of an IL-1α release inhibitor to said cell, thereby inhibiting IL-1α release from said cell.

2. The method of claim 1, wherein said release is stress-induced, and further wherein said IL-1α release inhibitor is selected from the group consisting of a copper chelator and a S100A13, or a fragment thereof.

3. The method of claim 3, wherein said S100A13 fragment is a S100A13ΔBR truncated protein.

4. The method of claim 4, wherein said copper chelator is tretrathiomolybdate (TTM).

5. A method of treating a condition mediated by stress-induced release of IL-1α from a cell, said method comprising administering an effective amount of a copper chelator to said cell, thereby treating said condition.

6. A method of inhibiting neointima formation following vessel injury in a mammal, said method comprising administering to said mammal an IL-lα release inhibiting amount of a copper chelator, thereby inhibiting said neointima formation.

7. A method of inhibiting macrophage infiltration following vessel injury in a mammal, said method comprising administering to said mammal an effective amount of a copper chelator, thereby inhibiting said macrophage infiltration.

8. The method of claim 7, wherein said macrophage infiltration is associated with inflammation.

9. A method of inhibiting cell proliferation associated with arterial wall injury, said method comprising administering an effective amount of a copper chelator to said mammal, thereby inhibiting said cell proliferation.

10. The method of claim 9, wherein said cell is a vascular smooth muscle cell and further wherein said copper chelator is TTM and said injury is caused by balloon angioplasty.

11. A method of inhibiting secretion of extracellular matrix following arterial wall injury in a mammal, said method comprising inhibiting non-traditional export of at least one of FGF-1 and IL-1α from a cell at the site of said injury, and further wherein said export is inhibited by administering an effective amount of a copper chelator to said mammal, thereby inhibiting said secretion of extracellular matrix in said mammal.

12. A method of inhibiting neointimal thickening associated with arterial wall injury in a mammal, said method comprising inhibiting non-traditional export of at least one of FGF-1 and IL-1α from a cell at the site of said injury, and further wherein said export is inhibited by administering an effective amount of a copper chelator to said mammal, thereby inhibiting said neointimal thickening in said mammal.

13. A method of inhibiting adventitial angiogenesis associated with arterial wall injury in a mammal, said method comprising inhibiting non-traditional export of at least one of FGF-1 and IL-1α from a cell at the site of said injury, and further wherein said export is inhibited by administering an effective amount of a copper chelator to said mammal, thereby inhibiting said adventitial angiogenesis in said mammal.

14. A method of identifying a compound useful for inhibiting adventitial angiogenesis associated with arterial wall injury in a mammal, said method comprising contacting a cell with a compound and comparing the level of release of a leader-less pro-inflammatory cytokine by said cell in response to temperature stress with the level of release of said cytokine from an otherwise identical cell not contacted with said compound in response to said temperature stress, wherein a decrease in said level of release of said leader-less pro-inflammatory cytokine by said contacted with said compound with said level of release of said cytokine from said otherwise identical cell not contacted with said compound is an indication that said compound inhibits said angiogenesis, thereby identifying a compound useful for inhibiting adventitial angiogenesis associated with arterial wall injury in a mammal.

15. The method of claim 14, wherein said leader-less pro-inflammatory cytokine is selected from the group consisting of FGF-1 and IL-1α.

16. A compound identified by the method of claim 14.

17. A kit for inhibiting release of IL-1α from a cell, said kit comprising an effective amount of an IL-1α release inhibitor, said kit further comprising an applicator and an instructional material for the use thereof.

18. A kit for treating a condition mediated by stress-induced release of IL-1α from a cell, said kit comprising an effective amount of a copper chelator, said kit further comprising an applicator and an instructional material for the use thereof.

19. A kit for inhibiting neointima formation following vessel injury in a mammal, said kit comprising an IL-1α release inhibiting amount of a copper chelator, said kit further comprising an applicator and an instructional material for the use thereof.

20. A kit for inhibiting restenosis following vessel injury in a mammal, said kit comprising an effective amount of a copper chelator, said kit further comprising an applicator and an instructional material for the use thereof.